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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/522,415	01/26/2005	Vivian I Teichberg	29147	5568

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EXAMINER	
GOUGH, TIFFANY MAUREEN	
ART UNIT	PAPER NUMBER
1657	
MAIL DATE	DELIVERY MODE
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/522,415	TEICHBERG, VIVIAN I	
	Examiner	Art Unit	
	Tiffany M. Gough	1657	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *11 June 2007*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-4,10-15,26 and 120-130* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-4,10-15,26 and 120-130* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Applicant's response filed 6/11/2007 has been received and entered into the case. New claims 128-130 have been entered into the instant application. Claims 1-4,10-15 26,120-130 are pending and have been considered on the merits. All arguments and amendments have been considered.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

The prior rejections under 35 USC § 112, second paragraph, are withdrawn in light of the claim amendments.

Claims 128-130 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Applicant does not distinctly claim what "...administering is effected to a peripheral blood..." means. It is not clear from the specification or claims what this means.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 1-4,10-15,120-127 stand rejected under 35 U.S.C. 103(a) as being unpatentable over Matthews et al (Journal of Neurochemistry, vol75, 2000) in view of http://www.bio.mtu/edu/campbell/bl4820/lectures/lec4/gotw41.htm,1996 and http://www.chem.qmul.ac.uk/iubmb/enzymeEC2/0601a/html.

Applicant claims a method of reducing extracellular brain glutamate levels by administering, to a subject in need of, an effective amount not exceeding 1g/kg body weight, of a naturally occurring or artificially modified glutamate modifying enzyme, specifically a transaminase and a co-factor of the enzyme.

Matthews et al teach the involvement of glutamate in neuronal death or injury associated with elevated extracellular glutamate levels in diseases such as ischemic brain injury, stroke, ALS, Parkinson's and Alzheimer's disease. They teach the enzymatic degradation of glutamate by GPT, which is useful in protecting the neurons from excitotoxic injury (see abstract). Their examples teach decreased levels of glutamate when incubated with GPT and its respective co-substrates and cofactors, pyridoxal phosphate and pyruvate. Results show glutamate degradation, therefore they expect GPT to provide useful protection against glutamate neurotoxicity associated with patients suffering from ischemic brain injury, stroke, ALS, Parkinson's and Alheimer's disease. Therefore, Matthews discloses the ability of GPT, a glutamate modifying enzyme, to decrease extracellular glutamate levels associated with the related disorders and diseases, i.e. a subject in need thereof.

Matthews do not teach administering to a subject in need thereof a glutamate modifying enzyme, however, they do teach diseases associated with elevated

extracellular glutamate levels and further teach a decrease in glutamate levels when incubated with a glutamate modifying enzyme GPT and it's respective substrates and co-factors. Thus, it would have been obvious to one of ordinary skill in the art, at the time of the claimed invention to administer a glutamate modifying enzyme to a subject in need thereof, such as one suffering from the diseases taught by Matthews associated with elevated extracellular glutamate levels because Matthews et al teach the GPT to be successful in decreasing elevated glutamate levels.

Moreover, at the time of the claimed invention, one of ordinary skill in the art would have been motivated to have administered a glutamate modifying enzyme to a subject in need thereof with a reasonable expectation for successfully decreasing extracellular glutamate levels because Matthews teach GPT to be useful in decreasing glutamate levels and useful in protecting against glutamate neurotoxicity associated with patients suffering from ischemic brain injury, stroke, ALS, Parkinson's and Alheimer's disease.

Matthews does not disclose the GPT to be artificially modified, however, protein engineering, i.e, modification, is well known in the art. For support see Haring et al , Protein Engineering, vol.15, 2002, who teach assembling artificial transaminases.

Further, Matthews does not teach administering GOT.

http://www.bio.mtu/edu/campbell/bl4820/lectures/lec4/gotw41.htm teaches pyridoxal phosphate to be a cofactor for GOT as well as the co-substrate oxaloacetate along with glutamate. Both enzymes GPT and GOT use glutamate as a substrate and pyridoxal phosphate as their cofactor to reversibly convert glutamate into 2-keto-

glutarate (see http://www.chem.qmul.ac.uk/iubmb/enzymeEC2/0601a/html). Thus, given what is taught by Matthews of GPT's ability to reduce glutamate levels and the similarities of GPT and GOT, i.e both glutamate converting enzymes using pyridoxal phosphate as their cofactor, differing only in their substrates pyruvate and oxaloacetate, it would have been obvious to one of ordinary skill in the art at the time of the invention to administer GOT, along with its respective cofactors and co-substrates, pyridoxal phosphate and oxaloacetate, to reduce extracellular glutamate levels in a subject in need given what is taught by Matthews et al of GPT's ability to reduce glutamate levels along with its respective cofactors and substrates.

Moreover, at the time of the claimed invention, one of ordinary skill in the art would have been motivated to have administered a glutamate modifying enzyme such as GOT to a subject in need thereof with a reasonable expectation for successfully decreasing extracellular glutamate levels because Matthews teach GPT to be useful in decreasing glutamate levels and useful in protecting against glutamate neurotoxicity associated with patients suffering from ischemic brain injury, stroke, ALS, Parkinson's and Alheimer's disease, further motivation is provided by the fact that both enzymes, GPT and GOT, use glutamate as a substrate and pyridoxal phosphate as their cofactor to reversibly convert glutamate into 2-keto-glutarate.

Response to Arguments

Applicant's arguments filed 6/11/2007 have been fully considered but they are not persuasive. Applicant argues that according to the instant invention extracellular brain glutamate levels can be reduced by the systemic administration of a glutamate

modifying enzyme to the peripheral blood of the subject intravenously thereby enhancing the brain to blood glutamate efflux. While these arguments have been considered they are not commensurate in scope with the instant claims. Such limitations are not claimed. Further applicant argues that Matthews does not administer but works with in vitro cultures of mouse cortices and that Matthews actually teaches away from in vivo use of glutamate modifying enzymes for treating glutamate related to neurotoxicity. Matthews teaches that the large molecular size of the enzyme is clearly precluded from crossing the blood brain barrier. Thus, Matthews teaches away from administering to the brain as well as its systemic administration through the peripheral circulation. Thus, as applicant admits and appears to urge, the method of the instant applicant is not enabled due to the enzymes large size. Applicants arguments are not persuasive.

Matthews clearly demonstrates that a glutamate modifying enzyme is highly active in degrading glutamate under physiologic conditions and is an effective neuroprotectant in cell culture models of exogenous and endogenous glutamate excitotoxicity (Discussion section). Thus, one of skill in the art **would** clearly have been motivated in contrary to applicants arguments, to administer a glutamate modifying enzyme to a subject with reasonable degree of success to protect against glutamate neurotoxicity. Further, Matthews suggests that because of the large molecular size of the enzymes, techiniques to disrupt the BBB, enhance BB trancytosis or bypasss the BBB would be needed to evaluate in vivo. Applicants method of claim 1 is open for further manipulation such as disruption of the BBB prior to administration of the enzyme, as it uses the language "comprising."

> The transitional term "comprising", which is synonymous with "including," "containing," or "characterized by," is inclusive or open-ended and does not exclude additional, unrecited elements or method steps. See, e.g., >*Mars Inc. v. H.J. Heinz Co.*, 377 F.3d 1369, 1376, 71 USPQ2d 1837, 1843 (Fed. Cir. 2004) ("like the term 'comprising,' the terms 'containing' and 'mixture' are open-ended.").< *Invitrogen Corp. v. Biocrest Mfg., L.P.*, 327 F.3d 1364, 1368, 66 USPQ2d 1631, 1634 (Fed. Cir. 2003) ("The transition 'comprising' in a method claim indicates that the claim is open-ended and allows for additional steps."); *Genentech, Inc. v. Chiron Corp.*, 112 F.3d 495, 501, 42 USPQ2d 1608, 1613 (Fed. Cir. 1997) ("Comprising" is a term of art used in claim language which means that the named elements are essential, but other elements may be added and still form a construct within the scope of the claim.); *Moleculon Research Corp. v. CBS, Inc.*, 793 F.2d 1261, 229 USPQ 805 (Fed. Cir. 1986); *In re Baxter*, 656 F.2d 679, 686, 210 USPQ 795, 803 (CCPA 1981); *Ex parte Davis*, 80 USPQ 448, 450 (Bd. App. 1948) ("comprising" leaves "the claim open for the inclusion of unspecified ingredients even in major amounts"). >In *Gillette Co. v. Energizer Holdings Inc.*, 405 F.3d 1367, 1371-73, 74 USPQ2d 1586, 1589-91 (Fed. Cir. 2005), the court held that a claim to "a safety razor blade unit comprising a guard, a cap, and a group of first, second, and third blades" encompasses razors with more than three blades because the transitional phrase "comprising" in the preamble and the phrase "group of" are presumptively open-ended. "The word 'comprising' transitioning from the preamble to the body signals that the entire claim is presumptively open-ended." *Id.* In contrast, the court noted the phrase "group consisting of" is a closed term, which is often used in claim drafting to signal a "Markush group" that is by its nature closed. *Id.* The court also emphasized that reference to "first," "second," and "third" blades in the claim was not used to show a serial or numerical limitation but instead was used to distinguish or identify the various members of the group. *Id.*<

Further the claims do not require a real subject or treating any disease state nor does applicant claim how the enzyme is actually administered.

Claims 1-4,10-15,26,120-125,128-130 stand rejected under 35 U.S.C. 103(a) as being unpatentable over WO 99/21565 in view of Matthews et al (Journal of Neurochemistry, vol75, 2000).and

http://www.bio.mtu/edu/campbell/bl4820/lectures/lec4/gotw41.htm,1996,

http://www.chem.qmul.ac.uk/iubmb/enzymeEC2/0601a/html,1976.

Applicant claims a method of reducing extracellular brain glutamate levels by administering, to a subject in need of, an effective amount not exceeding 1g/kg body weight, of a glutamate modifying enzyme and/or a co-factor of the enzyme.

WO 99/21565 discloses a method of treating individuals with disorders related to impaired mitochondrial and cerebral function. Such disorders include Huntington's disease which is a neurodegenerative disorder contributed by glutamate-induced neuronal death.

WO '565 discloses treating such disorders by administering a nutritional supplement/pharmaceutical composition in amounts of up to 15 g, containing a Kreb's cycle intermediate, such as oxaloacetic acid (oxaloacetate, a glutamate modifying enzyme co-factor) which is used to treat an individual with disorders related to impaired mitochondrial and cerebral function associated with glutamate excitotoxicity. Such disorders include Huntington's disease which is a neurodegenerative disorder contributed by glutamate-induced neuronal death.

http://www.bio.mtu/edu/campbell/bl4820/lectures/lec4/gotw41.htm teaches pyridoxal phosphate to be a cofactor for GOT as well as the co-substrate oxaloacetate along with glutamate. Both enzymes GPT and GOT use glutamate as a substrate and pyridoxal phosphate as their cofactor to reversibly convert glutamate into 2-keto-glutarate (see http://www.chem.qmul.ac.uk/iubmb/enzymeEC2/0601a/html). Thus, given what is taught by Matthews of GPT's ability to reduce glutamate levels and the

similarities of GPT and GOT, i.e both glutamate converting enzymes using pyridoxal phosphate as their cofactor, differing only in their substrates pyruvate and oxaloacetate, it would have been obvious to one of ordinary skill in the art at the time of the invention to administer GOT, along with its respective cofactors and co-substrates, pyridoxal phosphate and oxaloacetate, to reduce extracellular glutamate levels in a subject in need given what is taught by Matthews et al of GPT's ability to reduce glutamate levels along with its respective cofactors and substrates.

WO'565 does not teach administering a glutamate modifying enzyme, particularly an artificially modified enzyme, to a subject in need thereof.

However, as stated above Matthews et al teach the glutamate modifying enzyme, GPT to decrease glutamate levels when incubated along with its substrate and co-factors. They further suggest GPT to provide useful protection against glutamate neurotoxicity associated with patients suffering from ischemic brain injury, stroke, ALS, Parkinson's and Alheimer's disease. Further, protein engineering, i.e, modification, is well known in the art. For support see Haring et al, Protein Engineering, vol.15, 2002, who teach assembling artificial transaminases.

Given what is known in the art of the enzymatic reactions of GPT and GOT, it would have been obvious to one of ordinary skill in the art to administer a glutamate modifying enzyme, particularly GOT, it's cofactors and substrates, to a subject in need to reduce extracellular glutamate levels. Further, WO'565 teaches treating disorders associated with glutamate excitotoxicity by administering oxaloacetate, a glutamate modifying enzyme co-factor, in amounts of 15g, to a subject in need thereof.

Moreover, at the time of the claimed invention, one of ordinary skill in the art would have been motivated to have administered a glutamate modifying enzyme such as GOT, it's cofactors and substrates to a subject in need thereof with a reasonable expectation for successfully decreasing extracellular glutamate levels because Matthews teach GPT, it's cofactors and substrates to be useful in decreasing glutamate levels and useful in protecting against glutamate neurotoxicity associated with patients suffering from ischemic brain injury, stroke, ALS, Parkinson's and Alheimer's disease. WO'565 also teach treating those in need by administering the GOT cofactor/substrate oxaloacetate, thus reducing blood glutamate levels is intrinsic to the oxaloacetic acid/oxaloacetate. Thus, by practicing the method of WO '565 one would inherently be practicing the method as claimed. Further motivation is provided by the fact that both enzymes, GPT and GOT, use glutamate as a substrate and pyridoxal phosphate as their cofactor to reversibly convert glutamate into 2-keto-glutarate.

Claims 1-4,10,12-15,26,120,122,124,125 stand rejected under 35 U.S.C. 103(a) as being unpatentable over Matthews et al (Journal of Neurochemistry, vol75, 2000) in view of Geng et al (J. of Neurochemistry, vol. 68, no.6, 1997) and http://www.bio.mtu/edu/campbell/bl4820/lectures/lec4/gotw41.htm,1996, http://www.chem.qmul.ac.uk/iubmb/enzymeEC2/0601a/html,1976.

Applicant claims a method of reducing extracellular brain glutamate levels by administering, to a subject in need of, an effective amount not exceeding 1g/kg body weight, of a naturally occurring or artificially modified glutamate modifying enzyme, specifically a transaminase and a co-factor of the enzyme.

As stated above Matthews et al teach the glutamate modifying enzyme, GPT to decrease glutamate levels when incubated along with its substrate and co-factors. They further suggest GPT to provide useful protection against glutamate neurotoxicity associated with patients suffering from ischemic brain injury, stroke, ALS, Parkinson's and Alheimer's disease. Further, protein engineering, i.e, modification, is well known in the art. For support see Haring et al, Protein Engineering, vol.15, 2002, who teach assembling artificial transaminases.

Matthews does not teach administering GOT.

http://www.bio.mtu/edu/campbell/bl4820/lectures/lec4/gotw41.htm teaches pyridoxal phosphate to be a cofactor for GOT as well as the co-substrate oxaloacetate along with glutamate. Both enzymes GPT and GOT use glutamate as a substrate and pyridoxal phosphate as their cofactor to reversibly convert glutamate into 2-keto-glutarate (see http://www.chem.qmul.ac.uk/iubmb/enzymeEC2/0601a/html). Thus, given what is taught by Matthews of GPT's ability to reduce glutamate levels and the similarities of GPT and GOT, i.e both glutamate converting enzymes using pyridoxal phosphate as their cofactor, differing only in their substrates pyruvate and oxaloacetate, it would have been obvious to one of ordinary skill in the art at the time of the invention to administer GOT, along with its respective cofactors and co-substrates, pyridoxal phosphate and oxaloacetate, to reduce extracellular glutamate levels in a subject in need given what is taught by Matthews et al of GPT's ability to reduce glutamate levels along with its respective cofactors and substrates.

Geng teaches the administration of pyridoxal phosphate to epileptic patients. The increased glutamate levels, associated with elevated extracellular load of glutamate (see abstract), were normalized, thus reduced, by the administration of vitamin B6, i.e. pyridoxal phosphate (see p.2503, second paragraph) in amounts not exceeding 1g/kg (seep.2502).

Given what is known in the art of the enzymatic reactions of GPT and GOT, it would have been obvious to one of ordinary skill in the art to administer a glutamate modifying enzyme, particularly GOT, it's cofactors and substrates, to a subject in need to reduce extracellular glutamate levels. Further because Geng teaches the administration of pyridoxal phosphate to epileptic patients which normalized elevated extracellular load of glutamate (see abstract), it would be obvious to administer such co-factor along with it's respective glutamate converting enzyme, such as GOT.

Moreover, at the time of the claimed invention, one of ordinary skill in the art would have been motivated to have administered a glutamate modifying enzyme such as GOT, it's cofactors and substrates to a subject in need thereof with a reasonable expectation for successfully decreasing extracellular glutamate levels because Matthews teach GPT to be useful in decreasing glutamate levels and useful in protecting against glutamate neurotoxicity associated with patients suffering from ischemic brain injury, stroke, ALS, Parkinson's and Alzheimer's disease. Geng further teaches the administration of pyridoxal phosphate to epileptic patients, which normalized elevated extracellular load of glutamate (see abstract). Thus one would

have been motivated by the art to administer a glutamate modifying enzyme in combination with its respective cofactors and substrates.

Response to Arguments

Applicant's arguments filed 6/11/2007 have been fully considered but they are not persuasive. Applicant argues that Geng, like Matthews, teaches there are obstacles associated with using in vivo models,i.e. penetration though the BBB. Again, as stated above with respect the Matthews reference, applicant admits and appears to urge, the method of the instant applicant is not enabled due to the enzymes large size. Applicant argues that WO '565 does not teach administration of a glutamate modifying enzyme only administration of substrates and that applicant fails to understand how WO'565 teachings and Matthews can be combined to arrive at the present invention. While this is acknowledged, applicant is directed to their own specification, specifically examples 1 and 2 in which they merely add substrates pyruvate and oxaloacetate to blood cell fractions, further suggesting that adding the substrates the blood factions had the effect of decreasing concentrations of glutamate, further suggesting that once taken up intracellularly, transporters and substrates are able to activate intracellular GPT and GOT and that levels of whole blood glutamate can be manipulated in-vitro by the addition of GOT and/or GPT co-substrates. Further, there are no examples in applicants specification of actually administering to a subject the claimed glutamate modifying enzyme, only adding substrates and co-substrates to blood fractions. Given what is know in the art of administering pyruvate and/or oxaloacetate and their effect on neurotoxicity as well as the teachings of Matthews who clearly demonstrate that a

glutamate modifying enzyme is highly active in degrading glutamate under physiologic conditions and is an effective neuroprotectant in cell culture models of exogenous and endogenous glutamate excitotoxicity (Discussion section), one of skill in the art **would** clearly have been motivated in contrary to applicants arguments, to administer a glutamate modifying enzyme to a subject with reasonable degree of success to protect against glutamate neurotoxicity. Further, Wo'565 teahes administration of a substrate/co-factor in a supplement form which would inherently be treating the peripheral blood. Thus, applicants arguments are not persuasive.

Conclusion

No claims are allowed.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of

the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Tiffany M. Gough whose telephone number is 571-272-0697. The examiner can normally be reached on M-F 8-5 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jon Weber can be reached on 571-272-0925. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

SANDRA E. SAUCIER
PRIMARY EXAMINER